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### History and clinical signs

male king snake was referred to the ophthalmollogy service at the Western College of Veterinary Medicine with a history of progressive enlargement and cloudiness of the left eye of unknown duration. No previous therapy had been instituted. Physical examination revealed that the snake was in good bodily condition with no evidence of nasal discharge or oral cavity lesions. The neuro-ophthalmic examination revealed normal direct pupillary light reflexes. There were no menace or palpebral reflexes; these findings were normal for snakes, as they do not possess true eyelids. The consensual pupillary light and auriculopalpebral reflexes were absent in both eyes; these findings were also considered normal in snakes. The intraocular pressures, using applanation tonometry (Tonopen XL, Biorad Ophthalmic Division, Santa Clara, California), were 23 and 21 mm Hg in the right and left eyes, respectively. There was no fluorescein dye (Fluor-I-Strip AT, Ayerst Laboratories, Saint-Laurent, Quebec) uptake on either spectacle. Diffuse and focal biomicroscopy (Osram 64222, Carl Zeiss Canada, Don Mills, Ontario) confirmed diffuse bluish-white opacity of the left spectacle, and distention of the subspectacular space (area between spectacle and underlying cornea) with clear fluid (Figure 1). Both spectacles contained limbal-based vascularization and the right spectacle was transparent. The snake spectacle, formed embryologically by fusion of the eyelids, is normally vascularized. The anterior segments were normal bilaterally. Indirect ophthalmoscopy (Heine Omega 200, Heine Instruments Canada, Kitchener, Ontario) failed to reveal any posterior segment abnormalities of the right eye. The opacities in the spectacle precluded examination of the left fundus.

#### Discussion

Our clinical diagnosis was left bullous spectaculopathy due to occlusion of the nasolacrimal duct. The snake was sedated with ketamine hydrochloride (Vetalar, Vetrepharm Canada, London, Ontario) and midazolam (Versed, Hoffmann LaRoche, Mississauga, Ontario), intubated, and general anesthesia was maintained with isoflurane (IsoFlo, Abbott Laboratories, Saint-Laurent, Quebec). A thorough oral examination with biomicroscope failed to reveal any oral lesions. Skull radiographs were taken and no abnormalities were noted. The left spectacle was routinely prepared for surgery with copious lavages of dilute povidone-iodine (1:25). The snake was positioned in right lateral recumbency under the

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# What are your clinical diagnosis, diagnostic and treatment plans?



Figure 1. A king snake with bluish-white opacity of the left spectacle and marked distention of the subspectacular space.

operating microscope. A 30-gauge needle and tuberculin syringe were used to aspirate fluid from the subspectacular space. Smears of the fluid were made for cytologic examination, and fluid swabs were submitted for aerobic and anaerobic bacterial culture. A wedge from 5 to 6 o'clock was removed from the spectacle by using a No. 6500 Beaver blade (Beaver Mini-Blades, Becton Dickinson AcuteCare, Franklin Lakes, New Jersey, USA) and Wescott scissors. The subspectacular space was flushed with balanced salt solution (BSS Sterile Irrigating Solution, Alcon Canada, Mississauga, Ontario). The snake recovered from anesthesia uneventfully. The cytologic examination revealed clusters of degenerate heterophils with intracellular cocci, occasional epithelial cells, macrophages, and erythrocytes, as well as magenta-colored background material, suggestive of glycoproteins. Aerobic culture results confirmed the presence of Staphylococcus spp. The snake was treated with topical 0.3% ciprofloxacin (Ciloxan, Alcon Canada Inc., Mississauga, Ontario), 1 drop on the left eye, q6h, for 1 wk. Reexamination at 1 wk revealed decreased opacity of the left spectacle and no appreciable excess fluid accumulation in the subspectacular space. Reexamination at 2 mo revealed recurrence of the bullous spectaculopathy with accumulation of yellow, caseous material beneath the spectacle. Oral examination

was again normal. The left nasolacrimal duct was cannulated with a 27-gauge lacrimal cannula, and a retrograde flush with eye wash (Eye Stream, Alcon Canada) caused distention of the left spectacle and confirmed duct patency. A larger segment (approximately 1/8th) was exfoliated from the ventral part of the spectacle under topical anesthesia (0.5% proparacaine hydrochloride (Ophthetic, Allergan, Markham, Ontario) by using a No. 6500 Beaver blade. Topical therapy with 0.3% ciprofloxacin was administered at 1 drop, q6h for 1 wk, and then discontinued. Following the next ecdysis, the left spectacle appeared normal.

Bullous spectaculopathy, or pseudobuphthalmos, occurs in snakes due to occlusion of the nasolacrimal duct with subsequent distention of the subspectacular space with clear fluid secreted by the Harderian gland (1,2). Normally, the nasolacrimal duct exits the subspectacular space and enters the mouth, thereby resulting in direct communication between the subspectacular space and the oral cavity (1). Nasolacrimal duct blockage in snakes due to ulcerative stomatitis, oral or nasal neoplasia, brain granulomas, oral burns and scarring, cysts, congenital blockage, and idiopathic causes has been documented (1-4). Abscessation of the subspectacular space may result from ascension of an oral bacterium or fungus with occlusion of the nasolacrimal duct, a penetrating injury to the spectacle, or systemic infection (1,3,5,6). In this case, initial distention of the subspectacular space with clear fluid, given the absence of oral or nasal abnormalities, may have been due to an ascending staphylococcal dacryocystitis. The recurrence of the bullous spectaculopathy and abscessation following the first wedge resection likely occurred due to failure to remove a large enough section of spectacle and the subsequent inability of the topical antibiotics to penetrate the resealed spectacle.

Distention (fluid or abscess) of the subspectacular space is resolved by removing any oral obstructions or by draining the subspectacular space by incision of a 30° wedge in the ventral spectacle (1,6). Topical antibiotic or antibiotic/steroid solutions (if no infection) are used to prevent entry of infection until the spectacle seals (1,3,5,7). Cases of ulcerative stomatitis should be treated concurrently with systemic antibiotics and hydrogen peroxide mouthwashes (1,5,7). Occasionally, congenital and idiopathic cases resolve spontaneously (4).

#### References

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#### **MAY/MAI 2000**

American Animal Hospital Association — Implementing Behavioral Wellness Services Workshop. May 12–13, 2000 in Lakewood, Colorado, USA. Contact: Contact: AAHA Member Service Center, tel.: (800) 883-6301 or (303) 986-2800.

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641-0234; e-mail: ICE2000@ZEN-Inc.com; Web site: www.ZEN-Inc.com/ICE2000.

Saskatchewan Association of Veterinary Technologists — Mini-Conference and Annual General Meeting. May 13, 2000 in Regina, Saskatchewan. Contact: Leanne Malec, SAVT, tel.: (306) 665-6787; fax: (306) 665-6786.

Embryo Transfer Workshop. May 1-5, 2000 at the Western College of Veterinary Medicine. Contact: Dr. Reuben Mapletoft, Department of Herd Medicine and Theriogenology, Western College of Veterinary Medicine, University of Saskatchewan, 52 Campus Drive, Saskatoon, Saskatchewan 57N 5B4; tel.: (306) 966-7149; fax: (306) 966-7159.

Veterinary Homeopathy Course. May 18–21, 2000 in Tiberon, California, USA. Case-taking, repertory and materia medica, remedy selection, evaluating responses, managing acute and chronic cases, practical use of homeopathy in a clinical setting. Contact: Dr. David Evans, Natural Care Clinic for Pets, RR#3 Chester Basin, Nova Scotia B0J 1K0, tel.: (902) 275-3553, fax: (902) 275-2435.

American Animal Hospital Association — Veterinary Management Development School — Human Resource Intensive. May 19–20, 2000 in Boston, Massachusetts, USA. Contact: AAHA Member Service Center, tel.: (800) 883-6301 or (303) 986-2800.

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American Animal Hospital Association — AAHA Technician Academy. May 19–21, 2000 in Boston, Massachusetts, USA. Contact: AAHA Member Service Center, tel.: (800) 883-6301 or (303) 986-2800.